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CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
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=> biomarker and (urine or serum) and pulmonary
L1 91 FILE CAPLUS
L2 47 FILE MEDLINE
L3 38 FILE EMBASE
L4 137 FILE BIOSIS

TOTAL FOR ALL FILES
L5 313 BIOMARKER AND (URINE OR SERUM) AND PULMONARY

=> l5 and cadherin
L6 0 FILE CAPLUS
L7 0 FILE MEDLINE
L8 0 FILE EMBASE
L9 0 FILE BIOSIS

TOTAL FOR ALL FILES

```
L10           0 L5 AND CADHERIN

=> l5 and FEV1
L11           1 FILE CAPLUS
L12           2 FILE MEDLINE
L13           1 FILE EMBASE
L14           3 FILE BIOSIS

TOTAL FOR ALL FILES
L15           7 L5 AND FEV1

=> dup rem
ENTER L# LIST OR (END):l15
PROCESSING COMPLETED FOR L15
L16           4 DUP REM L15 (3 DUPLICATES REMOVED)

=> d l16 ibib abs total

L16  ANSWER 1 OF 4     MEDLINE                              DUPLICATE 1
ACCESSION NUMBER:    2002404352     MEDLINE
DOCUMENT NUMBER:     22148611   PubMed ID: 12153756
TITLE:               Environmental tobacco smoke exposure and pulmonary
                     function among adults in NHANES III: impact on the general
                     population and adults with current asthma.
AUTHOR:              Eisner Mark D
CORPORATE SOURCE:    Division of Occupational and Environmental Medicine,
                     Department of Medicine, University of California, San
                     Francisco, California, USA.. eisner@itsa.ucsf.edu
CONTRACT NUMBER:     K23 HL04201 (NHLBI)
SOURCE:              ENVIRONMENTAL HEALTH PERSPECTIVES, (2002 Aug) 110 (8)
                     765-70.
                     Journal code: 0330411. ISSN: 0091-6765.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200209
ENTRY DATE:          Entered STN: 20020803
                     Last Updated on STN: 20020925
                     Entered Medline: 20020924
AB   The impact of environmental tobacco smoke (ETS) exposure on adult
     pulmonary function has not been clearly determined. Because adults
     with asthma have chronic airway inflammation, they may be a particularly
     susceptible group. Using data from the Third National Health and Nutrition
     Examination Survey (NHANES III), I examined the cross-sectional
     relationship between serum cotinine, a biomarker of
     ETS exposure, and pulmonary function among 10,581 adult
     nonsmokers and 440 nonsmoking adults with asthma whose cotinine and
     spirometry data were available. I generated residuals, which are observed
     minus predicted values (based on Crapo equations), for forced expiratory
     volume in 1 sec (FEV1), forced vital capacity (FVC), and
     FEV1/FVC ratio to adjust for age, sex, and height. In addition, I
     used multivariate linear regression to control for sociodemographic
     characteristics and previous smoking history. Most adults with and without
     asthma had detectable serum cotinine levels, indicating recent
     ETS exposure (85.7% and 83.4%, respectively). Among nonsmoking male
     participants, I found no evidence that ETS exposure was related to
     decreased pulmonary function. In the nonsmoking female stratum,
     the highest cotinine tertile was associated with a lower FEV1
     [-100 mL; 95% confidence interval (CI), -143 to -56 mL], FVC (-119 mL; 95%
     CI, -168 to -69 mL), and FEV1/FVC ratio (-1.77%; 95% CI, -2.18%
     to -1.36%). Among women with asthma, the highest cotinine tertile was also
     associated with decreased FEV1 (-261 mL; 95% CI, -492 to -30
     mL), FVC (-291 mL; 95% CI, -601 to 20 mL), and FEV1/FVC ratio
```

(-1.6%; 95% CI, -3.3% to 0.19%). In conclusion, ETS exposure is associated with decreased **pulmonary** function in adult females, especially those with asthma. This analysis should provide further impetus for public policies that promote smoke-free environments.

L16 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:504373 BIOSIS
DOCUMENT NUMBER: PREV200200504373
TITLE: Involuntary smoking and asthma severity in children: Data from the Third National Health and Nutrition Examination Survey.
AUTHOR(S): Mannino, David M. (1); Homa, David M.; Redd, Stephen C.
CORPORATE SOURCE: (1) National Center for Environmental Health, Centers for Disease Control and Prevention, 1600 Clifton Rd, MS E-17, Atlanta, GA, 30333: dmannino@cdc.gov USA
SOURCE: Chest, (August, 2002) Vol. 122, No. 2, pp. 409-415. print. ISSN: 0012-3692.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Study objectives: We sought to determine the indicators of asthma severity among children in the United States with high and low levels of tobacco smoke exposure. Design: Cross-sectional study. Setting: Nationally representative survey of participants in the Third National Health and Nutrition Examination Survey (from 1988 to 1994). Participants: Five hundred twenty-three children with physician-diagnosed asthma. Measurements and results: We stratified the study participants into tertiles on the basis of **serum** levels of cotinine (a metabolite of nicotine that indicates tobacco smoke exposure). We used logistic and linear regression modeling, adjusting for known covariates, to determine the effect of high environmental tobacco smoke exposure on the following outcomes: asthma severity (determined using reported symptom and respiratory illness frequency); lung function; physician visits; and school absence. Among our study sample, 78.6% of children had mild asthma, 6.8% of children had moderate asthma, and 14.6% of children had severe asthma. Asthmatic children with high levels of smoke exposure, compared with those with low levels of exposure, were more likely to have moderate or severe asthma (odds ratio, 2.7 95% confidence interval (CI), 1.1 to 6.8) and decreased lung function, with a mean **FEV1** decrement of 213 mL or 8.1% (95% CI, -14.7 to -3.5). Conclusions: Involuntary smoke exposure is associated with increased asthma severity and worsened lung function in a nationally representative group of US children with asthma.

L16 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:859774 CAPLUS
DOCUMENT NUMBER: 136:353476
TITLE: Surfactant protein D (SP-D) and systemic scleroderma (SSc)
AUTHOR(S): Maeda, Manabu; Ichiki, Yoshiro; Aoyama, Yumi; Kitajima, Yasuo
CORPORATE SOURCE: Department of Dermatology, Gifu Prefectural Hospital, Gifu, 500-8226, Japan
SOURCE: Journal of Dermatology (2001), 28(9), 467-474 CODEN: JDMYAG; ISSN: 0385-2407
PUBLISHER: Japanese Dermatological Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors measured **serum** levels of SP-D in collagen diseases (110 cases) such as systemic scleroderma (SSc), scleroderma spectrum disorders (SSD), systemic lupus erythematosus (SLE), Sjogren syndrome (Sjs), dermatomyositis (DM), and rheumatoid arthritis (RA) and, as a control, dermatitis (DE) (109 cases). Addnl., the authors performed a correlation anal. to det. how these levels were related to **pulmonary** fibrosis and function test (vital capacity, %DLco). The **serum** levels of SP-D increased in SSc patients with Barnett type

III more than in SSc patients with Barnett type I or II, while they increased slightly in SSD (incomplete type of SSc) patients. The differences in these figures were statistically significant between the SSc (SSc & SSD) and non-SSc (SLE, DM, Sjs & RA) groups. The **serum** levels of SP-D in SSc patients with anti-topoisomerase I antibodies were statistically higher than those in SSc patients with other types of anti-nuclear antibodies. There was a statistically significant correlation between the severity of **pulmonary** fibrosis and the **serum** levels of SP-D, and a statistically neg. correlation between SP-D levels and vital capacity or %DLco, but there was no proportional correlation with the forced expiatory vol. (**FEV1**.0%). There was no statistical relation between pre- and post-therapy with photopheresis; however, there was a statistical correlation between the **serum** levels of SP-D and KL-6. In the group of collagen diseases, plasma levels of SP-D were higher than **serum** levels of SP-D. Patients with SSc possess higher levels of SP-D than do those with other collagen diseases and dermatitis, which may correspond to the severity of **pulmonary** fibrosis.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 4 OF 4 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000310683 MEDLINE
DOCUMENT NUMBER: 20310683 PubMed ID: 10853636
TITLE: Antioxidant nutrients and **pulmonary** function: the Third National Health and Nutrition Examination Survey (NHANES III).
AUTHOR: Hu G; Cassano P A
CORPORATE SOURCE: Division of Nutritional Sciences, Cornell University, Ithaca, NY 14853, USA.
CONTRACT NUMBER: HL 45731 (NHLBI)
SOURCE: AMERICAN JOURNAL OF EPIDEMIOLOGY, (2000 May 15) 151 (10) 975-81.
Journal code: 7910653. ISSN: 0002-9262.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000706
Last Updated on STN: 20000706
Entered Medline: 20000623

AB Recent studies of chronic obstructive **pulmonary** disease have raised interest in its relation to nutrition. Several dietary antioxidants have been positively associated with lung function in healthy, general population samples. This study considered the separate and joint effects of vitamin C, vitamin E, beta-carotene, and selenium intake and used both dietary assessment and **serum biomarkers** of antioxidant status. The authors used data from the Third National Health and Nutrition Examination Survey comprising a sample representative of the US population in 1988-1994 (n = 18,162 subjects aged > or =17 years). Multiple linear regression analysis examined the separate and joint effects of the antioxidants on the ratio of forced expiratory volume in the first second (**FEV1**)/height2 adjusted for covariates. Each of the dietary and **serum** antioxidant nutrients was significantly associated with **FEV1**. When they were considered simultaneously (dietary and **serum** variables considered in separate models), independent associations were observed for most nutrients. **Serum** beta-carotene was less positively associated with **FEV1** in smokers than nonsmokers, while **serum** selenium had a stronger positive association with **FEV1** in smokers. The authors found that higher levels of antioxidant nutrients are associated with better lung function. The finding that the antioxidants differ in both their overall association with lung function and in whether this association

varies by smoking status has implications for further research.

```
=> l5 and forced expiratory volume
L17           0 FILE CAPLUS
L18           4 FILE MEDLINE
L19           3 FILE EMBASE
L20           3 FILE BIOSIS

TOTAL FOR ALL FILES
L21          10 L5 AND FORCED EXPIRATORY VOLUME

=> dup rem
ENTER L# LIST OR (END):l21
PROCESSING COMPLETED FOR L21
L22            4 DUP REM L21 (6 DUPLICATES REMOVED)

=> l5 and adhesion
L23           2 FILE CAPLUS
L24           0 FILE MEDLINE
L25           0 FILE EMBASE
L26           0 FILE BIOSIS

TOTAL FOR ALL FILES
L27           2 L5 AND ADHESION

=> dup rem
ENTER L# LIST OR (END):l27
PROCESSING COMPLETED FOR L27
L28            2 DUP REM L27 (0 DUPLICATES REMOVED)

=> d l28 ibib abs total

L28  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2001:49174  CAPLUS
DOCUMENT NUMBER:          135:151556
TITLE:                    Elevated levels of soluble adhesion
                          molecules in sera and BAL fluid of
                          individuals infected with human T-cell lymphotropic
                          virus type 1
AUTHOR(S):                Seki, Masafumi; Higashiyama, Yasuhito; Kadota,
                          Jun-ichi; Mukae, Hiroshi; Yanagihara, Katsunori;
                          Tomono, Kazunori; Kohno, Shigeru
CORPORATE SOURCE:         Second Department of Internal Medicine, Nagasaki
                          University School of Medicine, Nagasaki, 852-8501,
                          Japan
SOURCE:                   Chest (2000), 118(6), 1754-1761
                          CODEN: CHETBF; ISSN: 0012-3692
PUBLISHER:                American College of Chest Physicians
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   T-lymphocytic alveolitis and increased levels of interleukin-2
     receptor-.alpha. (CD25)-bearing T cells in the BAL fluid (BALF) of human
     T-cell lymphotropic virus type 1 (HTLV-1) carriers have been reported.
     Several chemokines and adhesion mols. may contribute to the
     accumulation of T lymphocytes in the lungs of HTLV-1 carriers.  To clarify
     the correlation between adhesion mols. and HTLV-1-assocd.
     pulmonary disorders, the authors compared the distribution of
     T-lymphocyte subsets and sol. adhesion mols., including sol.
     intercellular adhesion mol. (sICAM)-1, sol. vascular cell
     adhesion mol.-1 (sVCAM-1), sol. L-selectin (sL-selectin), sol.
     E-selectin (sE-selectin), and sol. P-selectin (sP-selectin), in BALF and
     peripheral blood, between HTLV-1 carriers and noninfected healthy
     subjects.  Flow cytometric anal. with monoclonal antibodies to
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cell-surface antigens was used to identify T-lymphocyte subsets in BALF samples from HTLV-1 carriers and noninfected healthy control subjects. The levels of various sol. **adhesion** mols. in **serum** and in BALF were estd. by ELISA. Higher percentages of CD3+ cells, CD3-expressing human leukocyte antigen-DR antigen, and CD3+CD25+ cells were detected in the BALF of HTLV-1 carriers than in that of noninfected control subjects. The concns. of sICAM-1, sVCAM-1, sL-selectin, sE-selectin, and sP-selectin in the **sera** of patients were higher than in noninfected healthy control subjects. The concn. of sICAM-1 in the BALF of patients was higher than in noninfected healthy control subjects, and the concn. of sICAM-1 correlated well with the percentage of CD3+CD25+ cells. The concns. of **adhesion** mols. in the **sera** of and sICAM-1 in the BALF of HTLV-1 carriers were higher than those in noninfected individuals, and the concn. of sICAM-1 correlated well with the percentage of CD3+CD25+ cells in BALF. The authors' results suggest a possible interaction between activated T cells bearing CD25 and sol. **adhesion** mols., esp. sICAM-1, which may contribute to the **pulmonary** involvement in HTLV-1 carriers.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:6076 CAPLUS
DOCUMENT NUMBER: 134:26321
TITLE: A study of the pathogenesis in **pulmonary** asbestosis assessed by **serum** surfactant protein A and **serum** soluble intercellular **adhesion** molecule-1
AUTHOR(S): Okamoto, Yukinori
CORPORATE SOURCE: Second Dep. Intern. Med., Nara Med. Univ., Japan
SOURCE: Journal of Nara Medical Association (2000), 51(5), 257-268
CODEN: JNMAFJ
PUBLISHER: Nara Medical Association
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Surfactant protein A (SP-A) and intercellular **adhesion** mol.-1 (ICAM-1) were recently reported to be assocd. with fibrotic changes in **pulmonary** diseases. Inhaled asbestos induces **pulmonary** fibrosis leading to asbestosis. To evaluate the possible roles for the development of asbestosis, the **serum** levels of SP-A and sICAM-1 were measured and compared among workers exposed to asbestos (29 patients with asbestosis (PWAs), 50 asbestos-exposed workers without asbestosis (AEWs)), and 51 healthy controls (HCs). The mean **serum** levels of SP-A were significantly higher in PWAs (53.2.+-.33.4 ng/mL) and AEWs (45.1.+-.30.2 ng/mL) than that in HCs (28.8.+-.10.6 ng/mL). The mean **serum** levels of SP-A in smoking population of AEWs and HCs were significantly higher than those in non-smoking AEWs and HCs. The mean **serum** levels of SP-A in smokers were significantly higher in PWAs and AEWs than in HCs. The mean **serum** levels of SP-A in non-smokers were significantly higher in PWAs than in HCs. The mean **serum** levels of sol. ICAM-1 (sICAM-1) were significantly higher in PWAs (270.2.+-.114.8 ng/mL) and AEWs (250.7.+-.80.5 ng/mL) than in HCs (152.2.+-.49.9 ng/mL). The mean **serum** levels of sICAM-1 in AEWs were significantly higher in smokers than in non-smokers. The mean **serum** levels of sICAM-1 in smokers were significantly higher in PWAs and AEWs than in HCs. The mean **serum** levels of sICAM-1 in non-smokers were significantly higher in PWAs and AEWs than in HCs. The **serum** levels of SP-A were significantly correlated with the **serum** levels of sICAM-1 in PWAs and AEWs. In conclusion, the present study suggests that asbestos exposure is independently related to the elevation of **serum** levels of both SP-A and sICAM-1 and that this elevation is assocd. with the development of asbestosis. SP-A and sICAM-1 can be useful markers in detecting early asbestos-related

disorders.

```
=> s (pulmonary or lung) and (biomarker or marker) and (urine or serum)
L29        1128 FILE CAPLUS
L30        2887 FILE MEDLINE
L31        2236 FILE EMBASE
L32        2479 FILE BIOSIS

TOTAL FOR ALL FILES
L33        8730 (PULMONARY OR LUNG) AND (BIOMARKER OR MARKER) AND (URINE OR
                SERUM)

=> l33 and cadherin
L34           6 FILE CAPLUS
L35           4 FILE MEDLINE
L36           4 FILE EMBASE
L37           4 FILE BIOSIS

TOTAL FOR ALL FILES
L38          18 L33 AND CADHERIN

=> l38 and FEV1
L39           0 FILE CAPLUS
L40           0 FILE MEDLINE
L41           0 FILE EMBASE
L42           0 FILE BIOSIS

TOTAL FOR ALL FILES
L43           0 L38 AND FEV1

=> dup rem
ENTER L# LIST OR (END):l38
PROCESSING COMPLETED FOR L38
L44           12 DUP REM L38 (6 DUPLICATES REMOVED)

=> d 144 ibib abs total

L44  ANSWER 1 OF 12  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2002:716605  CAPLUS
DOCUMENT NUMBER:          137:213277
TITLE:                    Cell-based detection and differentiation of disease
                          states
INVENTOR(S):              Pressman, Norman J.; Hirsch, Kenneth S.
PATENT ASSIGNEE(S):       Monogen, Inc, USA; McCall, John, Douglas
SOURCE:                   PCT Int. Appl., 163 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.   DATE
     ---------------   ----  --------       ---------------   --------
     WO 2002073204      A2   20020919       WO 2002-GB1125    20020312
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
             CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
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PRIORITY APPLN. INFO.: US 2001-274638P P 20010312

AB The present invention is directed to a panel for detecting a generic disease state or discriminating between specific disease states using cell-based diagnosis. The panel comprises a plurality of probes each of which specifically binds to a **marker** assocd. with a generic or specific disease state, wherein the pattern of binding of the component probes of the panel to cells in a cytol. specimen is diagnostic of the presence or specific nature of said disease state. The present invention is also directed to a method of forming a panel for detecting a disease state or discriminating between disease states in a patient using cell-based diagnosis. The method involves detg. the sensitivity and specificity of binding of probes each of which specifically binds to a member of a library of **markers** assocd. with a disease state and selecting a limited plurality of said probes whose pattern of binding is diagnostic for the presence or specific nature of said disease state. The present method is also directed to a method of detecting a disease or discriminating between disease states comprising. The method involves contacting a cytol. sample suspected of contg. abnormal cells characteristic of a disease state with a panel according to claim 1 and detecting a pattern of binding of said probes that is diagnostic for the presence or specific nature of said disease state.

L44 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:172151 CAPLUS
DOCUMENT NUMBER: 136:230707
TITLE: Detection of aberrant promoter methylation by methylation-specific nested PCR in predicting cancer risk
INVENTOR(S): Belinsky, Steven A.; Palmisano, William A.
PATENT ASSIGNEE(S): Lovelace Respiratory Research Institute, USA
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002018649	A1	20020307	WO 2001-US26452	20010824
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001088379	A5	20020313	AU 2001-88379	20010824

PRIORITY APPLN. INFO.: US 2000-228057P P 20000825
WO 2001-US26452 W 20010824

AB A mol. **marker**-based method for monitoring and detecting cancer in humans. Aberrant methylation of gene promoters is a **marker** for cancer risk in humans. A two-stage, or "nested" polymerase chain reaction method is disclosed for detecting methylated DNA sequences at sufficiently high levels of sensitivity to permit cancer screening in biol. fluid samples, such as sputum, obtained non-invasively. The method is for detecting the aberrant methylation of the p16 gene, O 6 -methylguanine-DNA methyltransferase gene, Death-assocd. protein kinase gene, RAS-assocd. family 1 gene, or other gene promoters. The method offers a potentially powerful approach to population-based screening for the detection of **lung** and other cancers. Detection of one methylated allele in a mixt. contg. >50,000 methylated alleles is

demonstrated.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:937303 CAPLUS
DOCUMENT NUMBER: 138:20443
TITLE: Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
INVENTOR(S): Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 386 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002355079	A2	20021210	JP 2002-69354	20020313

PRIORITY APPLN. INFO.: JP 2001-73183 A 20010314
JP 2001-74993 A 20010315
JP 2001-102519 A 20010330

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises prepg. a nucleic acid sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample contg. the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose expression is altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-.beta. estradiol (E2), were found in mice by DNA chip anal.

L44 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:787469 CAPLUS
DOCUMENT NUMBER: 137:368150
TITLE: Preparation of recombinant MK-1/Ep-CAM and establishment of an ELISA system for determining soluble MK-1/Ep-CAM levels in **sera** of cancer patients
AUTHOR(S): Abe, Hironori; Kuroki, Motomu; Imakiire, Takayuki; Yamauchi, Yasushi; Yamada, Hiromi; Arakawa, Fumiko; Kuroki, Masahide
CORPORATE SOURCE: Department of Biochemistry, Fukuoka University School of Medicine, Fukuoka, 814-0180, Japan
SOURCE: Journal of Immunological Methods (2002), 270(2), 227-233
CODEN: JIMMBG; ISSN: 0022-1759
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The MK-1 antigen, also termed as Ep-CAM, is a membrane glycoprotein that is overexpressed on the majority of tumor cells of epithelial origin and thereby can be used as a target of immunodetection and immunotherapy of cancer. It has previously been shown that several type-I transmembrane proteins, including E-**cadherin**, ErbB-2 and intercellular adhesion mol.-1 (ICAM-1), may be useful as tumor **markers** because

they are released into the circulation of many cancer patients. To address the question of whether MK-1, the same type-I membrane protein, is also released into the **sera**, we developed a sandwich-type ELISA system by prepg. a recombinant MK-1 protein and two anti-MK-1 monoclonal antibodies with different epitope specificities. Using this ELISA, we found that the MK-1 levels in **serum** samples from healthy volunteers were all less than 2 ng/mL, whereas the Mk-1 levels in **sera** of about 10% of patients with malignant tumors of various tissue origins were increased to 2-78 ng/mL, indicating that MK-1 is released from tumor cells into the circulation under certain conditions. These findings should be borne in mind when trying to perform passive antibody therapy for cancer using anti-MK-1 antibody.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:863850 CAPLUS
DOCUMENT NUMBER: 136:32755
TITLE: Nucleic acid compositions, kits, and methods for identification, assessment, prevention, and therapy of human breast cancer
INVENTOR(S): Lillie, James; Palermo, Adam; Wang, Youzhen; Steinmann, Kathleen; Elias, Josh
PATENT ASSIGNEE(S): Millennium Predictive Medicine, Inc., USA
SOURCE: PCT Int. Appl., 2674 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001046697 A2		20010628	WO 2000-US35214	20001221

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR

PRIORITY APPLN. INFO.:
US 1999-PV171406 19991221
US 2000-PV176423 20000114
US 2000-PV190471 20000317
US 2000-PV193482 20000329
US 2000-PV205231 20000515
US 2000-PV213236 20000620
US 2000-PV219865 20000720

AB The invention relates to nucleic acid **marker** compns., kits and methods for detecting, characterizing, preventing, and treating human breast cancers. A variety of **markers** are provided, wherein changes in the levels of expression of one or more of the nucleic acid **markers** is correlated with the presence of breast cancer. The level of expression of numerous potential **markers** was measured in cells obtained from breast cancer tissue samples obtained form fifteen patients afflicted with breast cancer and from eleven breast cancer cell cultures, based on comparison with expression levels of each **marker** in corresponding non-cancerous breast tissue and cell cultures. The 15 cancer tissue samples include (i) five invasive lobular carcinomas (ILC), (ii) five invasive ductal carcinomas (IDC), and (iii) five samples of ductal carcinoma in situ (DCIS). As an addnl. evaluation of ability to indicate breast cancer, individual **markers** that were identified by transcriptional profiling criteria were also tested in six different subtracted library expts. In addn., protein profiling

expts. were undertaken to assess whether the proteins assocd. with the expression of individual **markers** of the invention are secreted. Table 21 lists approx. 43,500 GenBank Accession Nos. from the present invention. [This abstr. record is one of 8 records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

L44 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:338762 CAPLUS
DOCUMENT NUMBER: 134:362292
TITLE: Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile
INVENTOR(S): Farr, Spencer
PATENT ASSIGNEE(S): Phase-1 Molecular Toxicology, USA
SOURCE: PCT Int. Appl., 222 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001032928	A2	20010510	WO 2000-US30474	20001103
WO 2001032928	A3	20020725		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-165398P P 19991105
US 2000-196571P P 20000411

AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to det. the hypersensitivity of individuals to a given agent, such as drug or other chem., in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes assocd. with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes assocd. with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes assocd. with hypersensitivity. The expression of the genes predetd. to be assocd. with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and app. useful for identifying hypersensitivity in a subject are also disclosed.

L44 ANSWER 7 OF 12 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000487884 MEDLINE
DOCUMENT NUMBER: 20489842 PubMed ID: 11038142
TITLE: Androgen deprivation induces selective outgrowth of aggressive hormone-refractory prostate cancer clones expressing distinct cellular and molecular properties not present in parental androgen-dependent cancer cells.
COMMENT: Comment in: Cancer J. 2000 Jul-Aug;6(4):213-4

AUTHOR: Tso C L; McBride W H; Sun J; Patel B; Tsui K H; Paik S H; Gitlitz B; Caliliw R; van Ophoven A; Wu L; deKernion J; Belldegrun A
CORPORATE SOURCE: Department of Urology, Jonsson Comprehensive Cancer Center, University of California, Los Angeles, USA.
SOURCE: CANCER JOURNAL, (2000 Jul-Aug) 6 (4) 220-33.
Journal code: 100931981. ISSN: 1528-9117.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208
AB PURPOSE: The mechanism of progression of human prostate cancer (CaP) cells under androgen ablation therapy remains unclear. To study the alternative pathways of CaP cell growth under conditions of androgen deprivation, androgen-independent CaP variants were selected and expanded from an androgen-dependent CaP line via an in vitro androgen deprivation treatment. Cellular and molecular properties of these androgen-independent variants were characterized both in vitro and in vivo and compared with those of their parental androgen-dependent cells. METHODS: Androgen deprivation treatment of an androgen-dependent CaP cell line, LNCaP, was carried out by replacing culture medium with RPMI 1640 medium plus 10% charcoal-stripped **serum**. Cells that survived through the androgen deprivation treatment were harvested and expanded in the androgen-deficient culture medium and were designated CL-1. The CL-1 cells were also recultured in androgen-containing medium and designated CL-2. The growth (cell cycle analysis, 3H-thymidine incorporation assay, growth expansion, and colonization efficiency), expression of CaP-associated **markers** (semiquantitative reverse transcriptase polymerase chain reaction), interaction with endothelial and bone marrow stromal cells, sensitivity to anticancer agents and radiation (growth inhibition), and tumorigenicity of CL-1 and CL-2 cells were determined and compared with these characteristics in parental LNCaP cells. RESULTS: CL-1 and CL-2 cells are fast-growing cells when compared with parental LNCaP cells. They were capable of potentiating the growth of endothelial and bone marrow stromal cells in co-culture experiments and acquired significant resistance to radiation and to anticancer cytotoxic agents (Taxol paclitaxel, vinblastine, and etoposide). In contrast to the poorly tumorigenic parental LNCaP cells, CL-1 and CL-2 lines proved highly tumorigenic, exhibiting invasive and metastatic characteristics in intact and castrated mice or in female mice within a short period of 3 to 4 weeks. No growth supplements (e.g., Matrigel) were needed. When transfected with the green fluorescence protein (GFP) gene and transplanted orthotopically in the accessory sex gland, extensive metastatic disease from the primary CL tumor could be identified in bone, lymph nodes, **lung**, liver, spleen, kidney, and brain. Semiquantitative reverse transcriptase polymerase chain reaction analysis revealed a markedly distinct molecular expression profile in the CL lines: overexpression of basic fibroblast growth factor, interleukin-6, interleukin-8, vascular endothelial growth factor, transforming growth factor-beta, epidermal growth factor receptor, caveolin, and bcl-2 messenger RNAs and marked down-regulation of E-**cadherin**, p-53, and pentaerythritol tetranitrate. CONCLUSIONS: Early administration of hormonal therapy after failure of first-line treatment is associated with a profound clonal selection of aggressive AI variants, such as CL-1 and CL-2 lines. These tumor lines, with their parental counterparts, can serve as valuable tools for studying the cellular and molecular mechanisms of CaP progression and metastasis under hormonal therapy. CL-1 and CL-2 offer a unique and reproducible model for the evaluation of drug sensitivity and for other therapeutic modalities for advanced prostate cancer.

L44 ANSWER 8 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000405403 EMBASE
TITLE: Molecular **markers** for early cancer detection.
AUTHOR: Ahmed F.E.
CORPORATE SOURCE: F.E. Ahmed, Department of Radiation Oncology, Leo W. Jenkins Cancer Center, Brody School of Medicine, Greenville, NC 27858, United States
SOURCE: Journal of Environmental Science and Health - Part C Environmental Carcinogenesis and Ecotoxicology Reviews, (2000) 18/2 (75-125).
Refs: 357
ISSN: 1059-0501 CODEN: JSHREB
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
017 Public Health, Social Medicine and Epidemiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Uncontrolled cell proliferation is the hallmark of cancer, and tumor cells typically have acquired damage to genes that directly regulate their cell cycles (4). Interplay between the products of the cyclin D1, p16(INKA4) and retinoblastoma (Rb) susceptibility genes is involved in the regulation of cell cycle progression from G1 to S. Cell progression from G1 to S requires, among other things, activation of specific kinases, Cdks (Cdk4 and Cdk6), in association with cyclin D1. The active Cdk/cyclin D1 complex phosphorylates sequentially the Rb protein, thus releasing Rb-bound transcription factors of the E2F family. Free E2Fs transactivate genes that are essential for entry into S and DNA replication (S-phase effectors). Cdk/cyclin D1 activity is negatively regulated by binding of several cyclin kinase inhibitors, including p16(INKA4). The p16(INKA4) is believed to be activated in response to growth control stimuli through pathways which are not fully elucidated. Because pRb, cyclin D1 and p16(INKA4) are all upstream regulators of E2f activity, the frequent involvement of these proteins in human cancer suggests a central role for E2F in control of cell proliferation (9). Genetic alterations affecting p16(INKA4) and cyclin D1 protein that govern phosphorylation of the Rb protein and control exit from the G1 phase of the cell cycle, are so frequent in human cancers that inactivation of this pathway may well be necessary for tumor development. Like the p53 protein, component of this Rb pathway, although not essential for the cell per se, may participate in checkpoint functions that regulate homeostatic tissue renewal through life (10). It was recently recognized that the regulation of cell death (apoptosis) is also an important modulator of tumorigenesis. At least two genes linked to human cancers, bcl-2 and TP53, have been shown to regulate apoptosis. Cell culture studies have demonstrated that TP53 can induce, and bxl-2 suppress apoptosis in response to various stimuli. This raises the question as to how dysregulation of apoptosis contributes to neoplastic transformation and malignant cell growth (11). Genes for all these **markers** have been identified, isolated and sequenced, making it possible to apply molecular-based technologies for cancer detection. The continued acceleration of development of scientific information and new techniques necessitates the need for clinical application. Research in molecular genetics, cell biology, protein chemistry and immunology has identified many early changes occurring during neoplastic progression such as novel proteins, growth factors, cytokines, DNA damage and multiple genetic alterations. These changes present in tissue and other body fluids (e.g., plasma, **urine**, sputum, **lung** washings and feces) are now recognized as **markers** for impending cancer or for risk of cancer development. To explore fully the application of molecular profiles for earlier detection and risk assessment, it is essential to understand the molecular pathogenesis of cancer (i.e., the natural history of tumor progression) so that the biological behavior of an evolving lesion can be predicted with

Maryland 21287-6917, USA.
SOURCE: AMERICAN JOURNAL OF SURGICAL PATHOLOGY, (1998 Nov) 22 (11) 1393-403.
Journal code: 7707904. ISSN: 0147-5185.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981119

AB This report describes the clinicopathologic and immunohistochemical features of 14 cases of epithelioid trophoblastic tumor (ETT), a distinctive but rare gestational trophoblastic tumor. The patients with this neoplasm were in the reproductive age group and presented with abnormal vaginal bleeding. Although diagnosis was usually associated with a gestational event, the latter was sometimes remote. Two of the 14 patients presented with extrauterine ETT without evidence of prior gestational trophoblastic disease in the uterus. **Serum** human chorionic gonadotropin levels were elevated in eight of nine patients in whom this information was available. In the uterus, ETT presented as a discrete, hemorrhagic, solid and cystic lesion that was located either in the fundus, lower uterine segment, or endocervix. Microscopically, the tumor was composed of a relatively uniform population of mononucleate intermediate trophoblastic cells forming nests and solid masses. The cells resemble the trophoblastic cells in the chorion laeve, and we have therefore designated them "chorionic-type intermediate trophoblast." Typically, islands of trophoblastic cells were surrounded by extensive necrosis and were associated with a hyaline-like matrix creating a "geographic" pattern that is quite characteristic of this lesion. The mean mitotic count was two mitoses per 10 high-power fields, and the average Ki-67 nuclear labeling index was 18%. Immunohistochemically, all cases were diffusely positive for inhibin-alpha, cytokeratin (AE1/AE3), epithelial membrane antigen, E-**cadherin**, prolyl 4-hydroxylase, and epidermal growth factor receptor but were only focally immunoreactive for human placental lactogen, human chorionic gonadotropin, PlAP, and Mel-CAM. The monomorphic growth pattern of ETT resembles placental site trophoblastic tumor to a much greater degree than choriocarcinoma which is characterized by a dimorphic population of trophoblast. In contrast to placental site trophoblastic tumor, the cells of ETT are smaller and display less nuclear pleomorphism. In addition, ETT grows in a nodular fashion compared with the infiltrative pattern of placental site trophoblastic tumor. In some of the cases, the trophoblastic cells in ETT replaced the endocervical surface epithelium, giving the appearance that the tumor was derived from the cervix. Moreover, because the associated hyaline-like material in ETT resembles keratin, the tumor can be misinterpreted as a keratinizing squamous cell carcinoma of the cervix. Ten patients underwent total hysterectomy and two had an endometrial curettage only. The two patients who presented with extrauterine ETT underwent small bowel resection and **lung** resection. Two of 12 patients with ETT in the uterus developed metastasis in the **lungs** and bone. One of these patients is alive with disease at 43 months and one patient was lost to follow-up after 2 months. One of the two patients who had extrauterine disease died of widespread tumor 36 months after diagnosis. The remainder of the patients are alive and well from 1 to 120 months. In summary, ETT is a rare trophoblastic tumor that simulates carcinoma and can behave in a malignant fashion. It appears to be less aggressive than choriocarcinoma, more closely resembling the behavior of placental site trophoblastic tumor. Based on the morphologic and immunohistochemical features, it appears that ETT develops from neoplastic transformation of chorionic-type intermediate trophoblast.

L44 ANSWER 11 OF 12 MEDLINE DUPLICATE 3

(-1.6%; 95% CI, -3.3% to 0.19%). In conclusion, ETS exposure is associated with decreased **pulmonary** function in adult females, especially those with asthma. This analysis should provide further impetus for public policies that promote smoke-free environments.

L16 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:504373 BIOSIS
DOCUMENT NUMBER: PREV200200504373
TITLE: Involuntary smoking and asthma severity in children: Data from the Third National Health and Nutrition Examination Survey.
AUTHOR(S): Mannino, David M. (1); Homa, David M.; Redd, Stephen C.
CORPORATE SOURCE: (1) National Center for Environmental Health, Centers for Disease Control and Prevention, 1600 Clifton Rd, MS E-17, Atlanta, GA, 30333: dmannino@cdc.gov USA
SOURCE: Chest, (August, 2002) Vol. 122, No. 2, pp. 409-415. print. ISSN: 0012-3692.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Study objectives: We sought to determine the indicators of asthma severity among children in the United States with high and low levels of tobacco smoke exposure. Design: Cross-sectional study. Setting: Nationally representative survey of participants in the Third National Health and Nutrition Examination Survey (from 1988 to 1994). Participants: Five hundred twenty-three children with physician-diagnosed asthma. Measurements and results: We stratified the study participants into tertiles on the basis of **serum** levels of cotinine (a metabolite of nicotine that indicates tobacco smoke exposure). We used logistic and linear regression modeling, adjusting for known covariates, to determine the effect of high environmental tobacco smoke exposure on the following outcomes: asthma severity (determined using reported symptom and respiratory illness frequency); lung function; physician visits; and school absence. Among our study sample, 78.6% of children had mild asthma, 6.8% of children had moderate asthma, and 14.6% of children had severe asthma. Asthmatic children with high levels of smoke exposure, compared with those with low levels of exposure, were more likely to have moderate or severe asthma (odds ratio, 2.7 95% confidence interval (CI), 1.1 to 6.8) and decreased lung function, with a mean **FEV1** decrement of 213 mL or 8.1% (95% CI, -14.7 to -3.5). Conclusions: Involuntary smoke exposure is associated with increased asthma severity and worsened lung function in a nationally representative group of US children with asthma.

L16 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:859774 CAPLUS
DOCUMENT NUMBER: 136:353476
TITLE: Surfactant protein D (SP-D) and systemic scleroderma (SSc)
AUTHOR(S): Maeda, Manabu; Ichiki, Yoshiro; Aoyama, Yumi; Kitajima, Yasuo
CORPORATE SOURCE: Department of Dermatology, Gifu Prefectural Hospital, Gifu, 500-8226, Japan
SOURCE: Journal of Dermatology (2001), 28(9), 467-474 CODEN: JDMYAG; ISSN: 0385-2407
PUBLISHER: Japanese Dermatological Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors measured **serum** levels of SP-D in collagen diseases (110 cases) such as systemic scleroderma (SSc), scleroderma spectrum disorders (SSD), systemic lupus erythematosus (SLE), Sjogren syndrome (Sjs), dermatomyositis (DM), and rheumatoid arthritis (RA) and, as a control, dermatitis (DE) (109 cases). Addnl., the authors performed a correlation anal. to det. how these levels were related to **pulmonary** fibrosis and function test (vital capacity, %DLco). The **serum** levels of SP-D increased in SSc patients with Barnett type